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Published in:
Orthopaedic Journal of Sports Medicine

DOI:
10.1177/2325967119854324

Publication date:
2019

Document version
Publisher's PDF, also known as Version of record

Original Research

Gait Function and Postural Control 4.5 Years After Nonoperative Dynamic Treatment of Acute Achilles Tendon Ruptures

Merete B. Speedtsberg,*† MSc, Rasmus Kastoft,‡ MD, Kristoffer W. Barfod,‡§ MD, PhD, Jeanette Ø. Penny,‡|| MD, PhD, and Jesper Bencke,† MSc, PhD

Investigation performed at Human Movement Analysis Laboratory, Department of Orthopaedic Surgery, Copenhagen University Hospital Hvidovre, Hvidovre, Denmark

Background: An Achilles tendon rupture (ATR) is known to cause persistent biomechanical deficits such as decreased muscle strength in end-range plantar flexion and reduced tendon stiffness.

Purpose/Hypothesis: This study aimed to examine whether sustained asymmetries were present in dynamic stiffness and kinematic and kinetic variables in gait and single-leg balance at 4.5-year follow-up in conservatively treated patients recovering from an ATR. We hypothesized that patients who had recovered from ATRs exhibit a midterm increase in peak ankle dorsiflexion, a decrease in concentric work, and decreased dynamic stiffness during the stance phase of gait, along with increased single-leg standing sway in the injured leg compared with the uninjured leg.

Study Design: Case series; Level of evidence, 4.

Methods: This study was a cross-sectional medium-term follow-up of conservatively treated patients recovering from ATRs. A total of 34 patients who underwent nonoperative treatment were included for testing 4.5 years after a rupture. The Achilles tendon length was measured using ultrasound. Standard instrumented 3-dimensional (3D) gait analysis and single-leg standing balance were performed using 3D motion capture. Kinematic and kinetic ankle parameters were calculated during gait, and quasi-stiffness was calculated as the moment change per the change in the degree of dorsiflexion during the second (ankle) rocker of the gait cycle. Center of pressure displacement (sway length), along with rambling and trembling, was calculated for the single-leg balance task.

Results: Peak dorsiflexion in stance was 13.4% larger in the injured leg than the uninjured leg ($16.9^\circ \pm 3.1^\circ$ vs $14.9^\circ \pm 0.4^\circ$, respectively; $P \leq .001$). Peak dorsiflexion was not associated with the normalized Achilles tendon length (B = 0.052; $P = .775$). Total positive work in the plantar flexors was 23.9% greater in the uninjured leg than the injured leg (4.71 ± 1.60 vs 3.80 ± 0.79 J/kg, respectively; $P = .001$). Quasi-stiffness was greater in the uninjured leg than the injured leg during the initial (0.053 ± 0.022 vs 0.046 ± 0.020 N·m/kg/deg, respectively; $P = .009$) and late (0.162 ± 0.110 vs 0.139 ± 0.041 N·m/kg/deg, respectively; $P = .005$) phases of eccentric loading. No difference was found in sway length during single-leg stance between the injured and uninjured legs (1.45 ± 0.4 vs 1.44 ± 0.4 m, respectively; $P = .955$).

Conclusion: Patients treated conservatively have a small increase in peak dorsiflexion, decreased total concentric plantar flexor power, and decreased quasi-stiffness in initial and end-range dorsiflexion in the injured leg. These deviations could not be directly associated with the measured tendon elongation.

Registration: NCT02760784 (ClinicalTrials.gov).

Keywords: nonoperative dynamic treatment; Achilles tendon rupture; dynamic stiffness; balance

An acute Achilles tendon rupture (ATR) is a common injury among active adults at the age of 40 to 50 years, with a reported incidence ranging from 26.95 to 31.17 per 100,000 per year in Denmark.[14] An ATR is known to cause persistent elongation of the tendon after treatment, which is associated with biomechanical deficits such as decreased muscle strength in end-range plantar flexion and reduced tendon stiffness.[20] These functional deficits are reported regardless of the treatment modality for both short-term[2,15] and long-term outcomes.[16,24,26] The elongated tendon and decreased tendon stiffness may have implications for daily functional activities such as gait, running, and walking on stairs.[6,7]

Studies investigating early weightbearing have proposed it to have a beneficial effect on tendon healing in terms of increased tendon stiffness compared to standard

The Orthopaedic Journal of Sports Medicine, 7(6), 2325967119854324
DOI: 10.1177/2325967119854324

nonoperative regimens owing to the mechanical loading stimulus.[4,22] However, a randomized controlled study of nonoperatively treated ATRs did not find any difference in function after late or early weightbearing rehabilitation regimens at short-term[6] or 4.5-year follow-up.[17] A sustained deficit in the injured leg was observed in heel-rise work (limb symmetry index [LSI]) at short-term follow-up regardless of the nonoperative regimen.[17]

Reduced work output during the heel-rise test may not necessarily imply altered joint kinematics or dynamic joint stiffness. Dynamic stiffness comprises the total (active and passive) resistance of the plantar flexor–tendon complex to the angular movement of the ankle joint.[11] It involves passive stiffness along with possible active muscle contractions during movement and thereby includes muscle strength and possible co-contraction stabilization strategies during gait.[11] Reduced dynamic stiffness could result in decreased plantar flexor moments and lower power generation during gait as well as cause balance instabilities.

The present study aimed to examine if sustained elongation of the Achilles tendon was present in the injured leg 4.5 years after nonoperative treatment of an ATR and if, as a consequence, dynamic stiffness and kinematic and kinetic variables in gait and balance were different between the injured and uninjured legs. We hypothesized that patients recovering from ATRs exhibit a midterm increase in peak ankle dorsiflexion, a decrease in concentric work, and decreased dynamic stiffness during the stance phase of gait along with increased 1-leg standing sway in the injured leg compared to the uninjured leg. We further hypothesized that these kinematic and kinetic differences are associated with the Achilles tendon length.

METHODS

The present study was a cross-sectional medium-term follow-up of nonoperatively treated patients recovering from ATRs. The study was designed as an add-on to a randomized controlled trial (RCT) conducted from 2011 to 2013 that investigated the short-term effects of early weightbearing compared with nonweightbearing in nonoperatively treated ATRs.[6] This midterm follow-up was conducted in accordance with Consolidated Standards of Reporting Trials (CONSORT) guidelines and was approved by the Institutional Review Board of the Capital Region of Copenhagen, Denmark.[17] Follow-up of the primary outcome has been published previous to this work.[17]

Participants

Patients for the medium-term (4.5-year) follow-up were recruited from the 56 patients who were included in and completed the initial RCT previously described by Barfod et al.[6,7] A full description of initial and medium-term inclusion has been published by Kastoft et al.[17] Patients who suffered an additional new and significant lower limb injury such as cruciate ligament or tendon ruptures were excluded. Seven patients were excluded because of a new injury, 3 were excluded because of a rerupture succeeded by surgical treatment, and 12 declined the invitation or did not respond. Thus, 34 patients were included and pooled as the nonoperatively treated group for the medium-term follow-up (18 treated nonoperatively with early weightbearing and 16 treated with conventional nonoperative treatment).

The initial treatment has previously been described in detail.[6,17] Briefly, it consisted of standard nonoperative treatment, with immobilization for 8 weeks aimed to flex the ankle in the equinus position (20°-30° of plantar flexion) and controlled early motion after 2 weeks. A standardized rehabilitation protocol was followed 3 times per week for 1 hour from weeks 9 to 16. In daily activities, cycling was allowed from week 10 and jogging from week 14; however, these recommendations were individualized. Sport activities could be resumed after 6 months, but the patients were advised not to resume racquet or contact sports before 12 months. The treatment protocol for the patients included in this cross-sectional medium-term follow-up differed only in the permission to bear weight, in which 18 patients were allowed full weightbearing from day 1 and the remaining 16 after 6 weeks.[6,7]

Procedure and Data Processing

All tests and measurements were carried out by a medically trained test manager who was blinded to the patients' initial RCT allocation. Blinding of the injured versus uninjured leg was not possible because of visually apparent differences. Before participation, the patients completed the Achilles tendon Total Rupture Score (ATRS).[21] The Achilles tendon length was measured using a previously validated and well-described ultrasound-based technique, with a minimal detectable change of 10 mm.[8] The tendon length was measured as the distance from the calcaneus to the medial head of the gastrocnemius muscle, with the patient in a prone position, the ankles resting at 10° of plantar flexion on a foam roll, and the knees flexed at 10° to 20°. The distal landmark was defined as the posterosuperior

*Address correspondence to Merete B. Speedtsberg, MSc, Human Movement Analysis Laboratory, Department of Orthopaedic Surgery, Copenhagen University Hospital Hvidovre, Kettegård Alle 30, 2650 Hvidovre, Denmark (email: merete.brink.speedtsberg@regionh.dk).
†Human Movement Analysis Laboratory, Department of Orthopaedic Surgery, Copenhagen University Hospital Hvidovre, Hvidovre, Denmark.
‡Clinical Orthopaedic Research Hvidovre, Department of Orthopaedic Surgery, Copenhagen University Hospital Hvidovre, Hvidovre, Denmark.
§Sports Orthopaedic Research CenterCopenhagen, Department of Orthopaedic Surgery, Copenhagen University Hospital Hvidovre, Hvidovre, Denmark.
||Section of Foot and Ankle Orthopaedics, Department of Orthopaedic Surgery, Zealand University Hospital, Kge, Denmark.

The authors have declared that there are no conflicts of interest in the authorship and publication of this contribution. AOSSM checks author disclosures against the Open Payments Database (OPD). AOSSM has not conducted an independent investigation on the OPD and disclaims any liability or responsibility relating thereto.
Ethical approval for this study was obtained from the Institutional Review Board of the Capital Region of Copenhagen, Denmark (reference No. 16015461).

TABLE 1
Anthropometric Measurements and Patient Characteristics (N = 34)[a]

	Mean ± SD
Age, y	45 ± 7
Height, m	1.77 ± 0.07
Weight, kg	88.6 ± 14.4
Achilles tendon Total Rupture Score	83.2 ± 16.9
Tendon length, cm	
Uninjured	18.8 ± 2.2
Injured	20.5 ± 1.8
Passive dorsiflexion, deg	
Uninjured	11.0 ± 5.3
Injured	12.9 ± 5.0
Calf circumference, cm	
Uninjured	40.0 ± 2.9
Injured	38.3 ± 3.2

corner of the calcaneus, and the proximal landmark was defined as the distal-most muscle fibers of the medial head of the gastrocnemius. Measurements of the tendon length were supervised by an experienced radiologist. The Achilles tendon length was normalized to height for statistical analysis but was reported as the absolute length for anthropometric measurements (Table 1).

The calf circumference was measured 13 cm below the distal tip of the patella with the patient sitting on the examination bench and the feet hanging off the side. The calf circumference was normalized to body weight for statistical analysis but was reported as the absolute circumference for anthropometric measurements (Table 1).

Standard instrumented 3-dimensional (3D) gait analysis and 1-leg standing balance were performed using 3D motion capture with 8 T40 cameras (Vicon Motion Systems) and 2 OR6-7 force plates (AMTI).

Gait Analysis

Twenty-two reflective skin markers were placed over anatomic bony landmarks according to the modified Plug-in Gait model (Vicon Motion Systems).[23] Thigh markers were placed on the patella to minimize the effect of wobbling masses,[27] and markers were placed on the iliac crest to ensure redundancy of markers. The patients were instructed to walk barefoot at a self-selected speed on a 10-m level walkway. This was continued until 5 gait trials for each leg with complete hits on the force plates were obtained. The position of each marker was recorded 3-dimensionally at 100 Hz, and ground-reaction forces were sampled at 1000 Hz. Kinematic and kinetic data from gait analysis were subsequently calculated using inherent software (Nexus 2.5; Vicon Motion Systems). The raw kinematic data were in this process filtered using a Woltring cubic spline filter. The discrete outcome parameters were derived using custom-written MATLAB script (MATLAB 8.5.0; MathWorks). The mean of the 5 obtained gait trials on each leg was used for further statistical analysis.

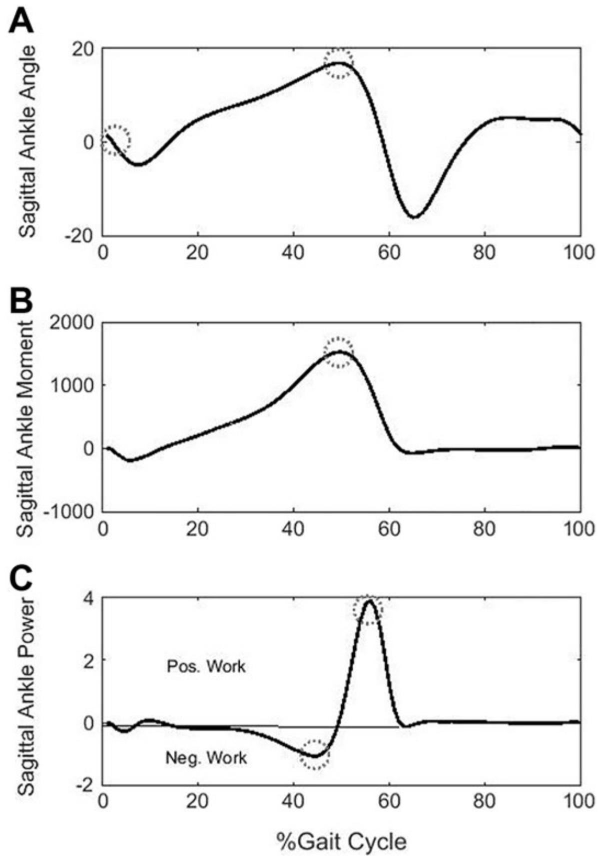

Figure 1. Graphs showing selected kinematic and kinetic outcome parameters. (A) The sagittal ankle angle curve with peak angle in the stance phase and angle at initial contact (IC). (B) The sagittal ankle moment curve with peak sagittal ankle moments. (C) The sagittal ankle power curve with peak negative (Neg.) power and peak positive (Pos.) power as well as the distinction between total negative work and total positive work.

To describe the kinematic differences between the injured and uninjured legs during gait, the sagittal-plane ankle angle (in degrees) was calculated at initial contact and at peak dorsiflexion during the stance phase (Figure 1). The kinetic differences were described by extracting peak plantar flexor moment (N·m/kg), peak eccentric (negative) power (W/kg), and peak concentric (positive) power during the stance phase as well as the instant of these peaks from the time of heel strike (in seconds). Total internal plantar flexor moment (plantar flexor angular impulse) was calculated by numerical integration of the moment curve as the cumulative sum of positive moments. Total negative and positive work produced during the stance phase were calculated by numerical integration of the power curves. Eccentric work was calculated as the cumulative sum of negative power generation and concentric work as the cumulative sum of positive power generation during the stance phase (in J/kg).

Dynamic stiffness of the plantar flexor–tendon complex was expressed as quasi-stiffness[11] and was calculated for

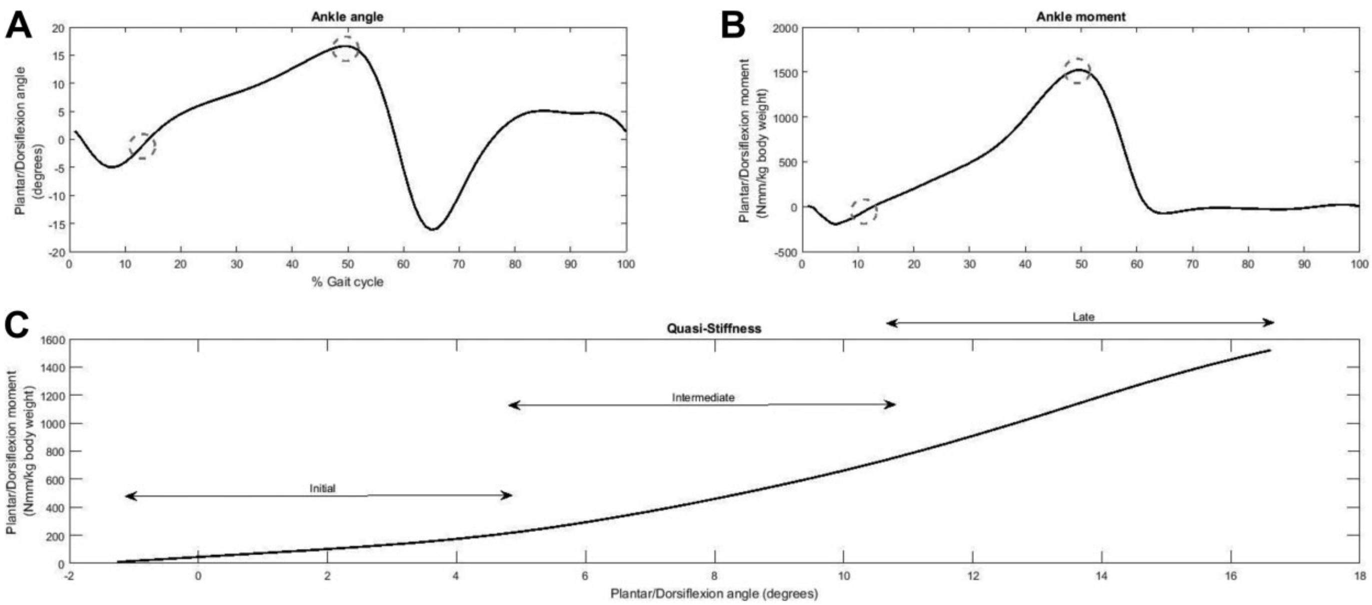

Figure 2. Quasi-stiffness is calculated as the rise in plantar flexor moment as a function of the change in dorsiflexion during the second rocker. The second rocker is indicated in the (A) ankle angle and (B) ankle moment curves. (C) Quasi-stiffness of initial, intermediate, and late phases of the second rocker.

3 separate phases during eccentric loading of the plantar flexors (second rocker of the gait cycle) (Figure 2). The period from initiation of the internal plantar flexor moment until the time of peak plantar flexor moment was selected, and stiffness was calculated as the change in plantar flexor moment (in N·m/kg body mass) per change in the dorsiflexion angle (in degrees). The whole period of the joint moment-to-angle curves was divided into 3 equally long periods, and first-order polynomial fitting was used to obtain the slope coefficient within each of the 3 (initial, intermediate, late) periods during eccentric loading of the plantar flexors during gait.

One-Leg Standing Balance

The degree of postural balance was quantified during the 1-leg standing balance task. The patients were instructed to perform the task positioned on a force platform with the feet aligned along the anteroposterior axis of the platform; patients were instructed to stand as still as possible, with the arms crossed across the chest and the head in a fixed position. The task was performed 3 times for 30 seconds each on both the injured and uninjured legs, alternating between legs. Each patient was given a short 30-second break between trials, including a walk of a preset 6-m distance to prevent the disturbance of balance due to the hydrostatic accumulation of blood in the lower leg. The ground-reaction force was recorded at 1000 Hz when the patients indicated that a stable 1-leg position had been achieved.

Balance differences between the injured and uninjured legs were expressed as sway length along with rambling and trembling.[30] Calculations were performed using custom-written MATLAB script (MATLAB 8.5.0). Force plate data were down-sampled to 100 Hz before smoothing the anteroposterior (AP) and mediolateral (ML) components of the center of pressure (CoP) using a fourth-order low-pass Butterworth filter with a cut-off frequency of 10 Hz. The distance traveled of the CoP was calculated as sway length using the Pythagorean theorem. Rambling-trembling analysis was performed as proposed by Zatsiorsky and Duarte.[30] This analysis decomposes the CoP trajectory into "rambling" and "trembling" contributions to postural control, enabling a distinction between supraspinal motor cortex–controlled oscillations of the CoP (rambling) and the effect of peripheral proprioceptive feedback (trembling).

To estimate the rambling trajectory, the instant equilibrium positions were identified as the CoP positions when the horizontal force Fhor = 0 and interpolated using a cubic spline function. To obtain the trembling trajectory, deviations of the CoP trajectory from the interpolated instant equilibrium positions were determined.[29,30] Rambling and trembling trajectories were quantified by the standard deviation in the AP and ML directions. The mean of the 2 best trials (shortest sway length) on each leg was used for further statistical analysis.

Statistical Analysis

Differences between the injured and uninjured limbs in gait kinematics and kinetics along with sway parameters were analyzed using a paired t test. Differences between injured and uninjured limbs in quasi-stiffness from gait analysis were analyzed using the Wilcoxon signed-rank test. Associations between the normalized Achilles tendon length and normalized calf circumference and functionally relevant gait parameters were analyzed using linear

TABLE 2
Kinematic, Kinetic, and Quasi-Stiffness Results From Gait Analysis[a]

	Uninjured	Injured	P
Kinematics			
Peak dorsiflexion, deg	14.9 ± 0.4	16.9 ± 3.1	≤.001[b]
Dorsiflexion at initial contact, deg	0.2 ± 2.3	1.3 ± 2.3	.017[b]
Kinetics			
Plantar flexor impulse, N·m*s/kg	39.19 ± 5.62	37.32 ± 4.99	.003[b]
Total positive plantar flexor work, J/kg	4.71 ± 1.60	3.80 ± 0.79	.001[b]
Peak positive plantar flexor moment, N·m/kg	1.53 ± 0.16	1.55 ± 0.17	.271
Peak positive plantar flexor power, W/kg	4.39 ± 0.88	4.30 ± 0.75	.479
Total negative plantar flexor work, J/kg	–2.20 ± 0.49	–2.31 ± 0.44	.179
Peak negative plantar flexor moment, N·m/kg	0.22 ± 0.05	0.20 ± 0.05	.028[b]
Peak negative plantar flexor power, W/kg	0.99 ± 0.34	1.18 ± 0.33	≤.001[b]
Quasi-stiffness, N·m/kg/deg			
Initial phase	0.053 ± 0.022	0.046 ± 0.020	.009[b]
Intermediate phase	0.104 ± 0.091	0.102 ± 0.043	.106
Late phase	0.162 ± 0.110	0.139 ± 0.041	.005[b]

[a]Data are presented as mean ± SD.
[b]Statistically significant difference between uninjured and injured legs ($P < .05$).

regression models. Associations between quasi-stiffness and sway length were analyzed using linear regression models. All models were adjusted for early weightbearing versus nonweightbearing because of unexpected differences in the pooled nonoperatively treated population. The level of significance was set at $P < .05$, and results are reported as mean ± SD for normally distributed data. Stiffness data were not normally distributed and are reported as the median. Regression coefficients are reported as B.

RESULTS

The physical examination showed clinical differences between the injured and uninjured legs; these are presented in Table 1 along with an overview of participant characteristics.

Gait: Kinetics and Kinematics

Significant differences in ankle joint kinematics and kinetics and the timing of power production were found between the uninjured and injured legs at follow-up after 4.5 years. These differences are summarized in Table 2.

During gait, peak dorsiflexion in stance was, on average, 13.4% larger in the injured leg than the uninjured leg ($P \leq .001$). At initial contact, the difference between the legs was significant but smaller ($P = .017$). Peak dorsiflexion was not associated with the normalized Achilles tendon length (B = 0.052; $P = .775$).

Plantar flexor impulse (total summated internal plantar flexor moment) was significantly higher in the uninjured leg compared to the injured leg ($P = .003$), and total positive work in the plantar flexors was 23.9% higher in the uninjured leg compared to the injured leg ($P = .001$). Peak eccentric (negative) power was larger in the injured leg and occurred, on average, 29 milliseconds later in the stance phase ($P = .002$). No difference was found for total negative work. No statistically significant associations were found between early weightbearing and peak power, minimum power, or total negative and positive work.

Gait: Quasi-Stiffness

Quasi-stiffness was calculated during initial, intermediate, and late eccentric loading (Figure 3). Quasi-stiffness was significantly higher in the uninjured leg than the injured leg during the initial and late phases of eccentric loading ($P = .009$ and .005, respectively). No statistical difference was found in the intermediate phase. Quasi-stiffness in the initial phase of eccentric loading was associated with plantar flexor impulse in the injured leg, and quasi-stiffness in the intermediate phase was associated with total negative work in the injured leg. In the uninjured leg, quasi-stiffness in the intermediate phase was associated with peak positive power and total positive work. Finally, in the late phase, it was associated with total positive work and peak negative power in both legs (Table 3). Quasi-stiffness in the injured leg was not associated with the normalized Achilles tendon length (initial: $P = .672$; intermediate: $P = .695$; late: $P = .802$) or early weightbearing (initial: $P = .177$; intermediate: $P = .779$; late: $P = .821$).

Static Balance

No difference was found in sway length during 1-leg stance between the injured and uninjured legs, nor did the decomposition of CoP displacement into rambling and trembling reveal any differences in either the AP or ML direction (Table 4). Linear regression analyses showed no association between sway length in the injured leg and the ATRS (B = 0.00; $P = .68$). For the injured leg, quasi-stiffness in initial eccentric loading was associated with an estimated 0.075-m increase in sway length per 0.01-N·m/kg/deg increase in stiffness (B = 0.075; $P = .0271$). This association was, however, not significant in the uninjured leg (B = 0.056; $P = .0709$).

DISCUSSION

In the current study, we showed a 2.0° (+13.4% in the injured leg) increased peak dorsiflexion during gait in the injured leg compared to the uninjured leg 4.5 years after ATR. The increased range of motion was accompanied by decreased functional stiffness in the initial and late parts of eccentric loading (second rocker), decreased total positive work and plantar flexor impulse, and

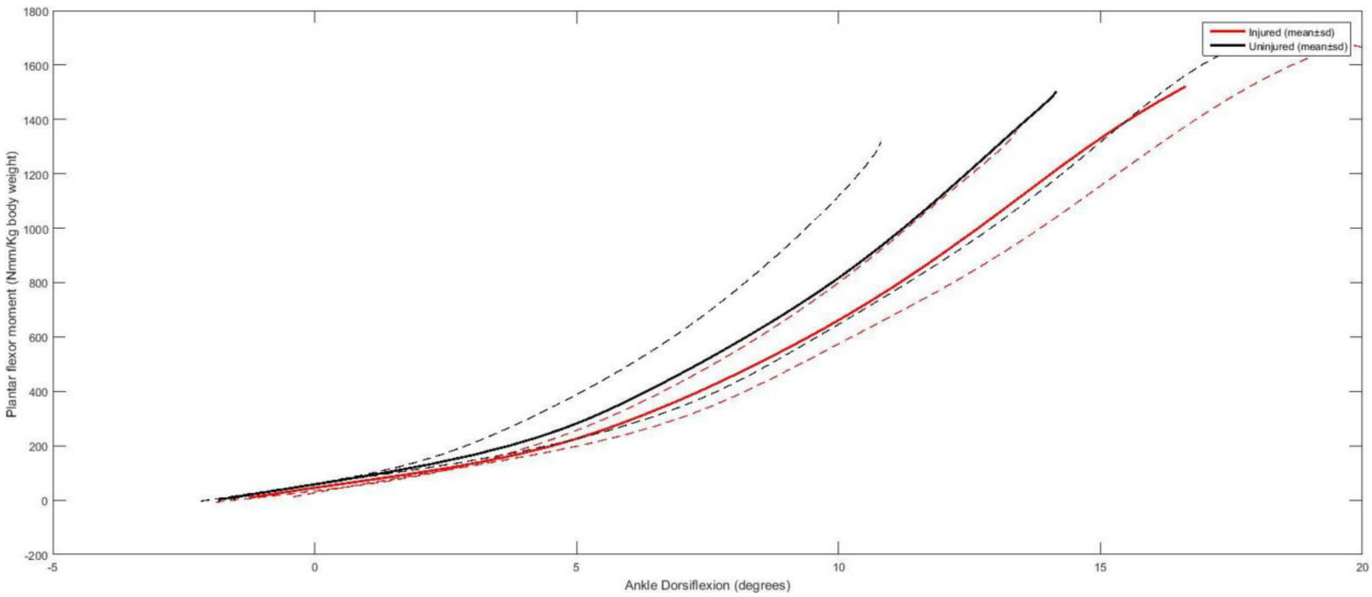

Figure 3. Quasi-stiffness of the uninjured and injured legs during gait. The group means are represented as solid lines ± 1 SD (broken lines).

TABLE 3
Statistically Significant Associations Between Quasi-Stiffness and Kinematic and Kinetic Parameters From Regression Models

	Initial Phase		Intermediate Phase		Late Phase	
	Injured	Uninjured	Injured	Uninjured	Injured	Uninjured
Peak positive moment					B = 2650.44; $P < .001$	
Peak positive power				B = 3.30; $P = .035$		
Peak negative power					B = 3.7; $P = .006$	B = 2.4; $P < .001$
Total positive work				B = –5.75; $P = .043$	B = 9.6; $P = .005$	B = 9.5; $P = .004$
Total negative work			B = 4.65; $P = .016$			B = 3.1; $P = .002$
Plantar flexor impulse	B = 87702.34; $P = .030$				B = 65155.56; $P = .001$	

TABLE 4
Sway Length and Rambling/Trembling During 1-Leg Standing Balance Task[a]

	Uninjured	Injured	*P*
Sway length, m	1.44 ± 0.4	1.45 ± 0.4	.955
Anteroposterior rambling	7.0 ± 1.8	6.7 ± 1.6	.414
Anteroposterior trembling	3.2 ± 0.7	3.3 ± 0.9	.710
Mediolateral rambling	5.3 ± 1.4	5.4 ± 1.2	.908
Mediolateral trembling	3.3 ± 0.9	3.5 ± 0.9	.123

[a]Data are presented as mean ± SD.

increased peak eccentric (negative) power for the injured leg compared to the uninjured leg. No statistically significant differences were found in 1-leg standing balance. In general, the normalized Achilles tendon length and normalized calf circumference were not statistically associated with any of the functional outcomes from gait and standing balance.

Gait Kinematics and Kinetics

Increased peak dorsiflexion for the injured leg, as found in the present study, has also been observed in other studies. An earlier midterm follow-up on nonsurgically treated patients reported a similar level of side-to-side differences, with peak passive dorsiflexion that was 7.5% (1°) larger in the injured leg after 2 to 4 years.[24] A difference of 2° may not seem clinically relevant, and the ATRS value (83.2) did indicate that this group of patients may have recovered fairly well. We did, however, observe corresponding kinetic differences that may indicate or reveal functional consequences or causes for this difference.

Interestingly, the present study found no significant association between the normalized Achilles tendon length and peak dorsiflexion. Sustained lengthening of the Achilles tendon in the injured leg is a well-established observation after a rupture,[16,22] but only a few other studies have investigated the association between ultrasound-measured Achilles tendon length and gait kinematics and kinetics. Brorsson and colleagues[10] investigated this association and found a weak to moderate correlation between the Achilles tendon length and gait kinematics. An alternative explanation could be found in the fact that forward progression of the tibia in the second rocker is controlled by eccentric contraction of the plantar flexors. Increased peak dorsiflexion may therefore reveal a sustained deficit in eccentric dynamic control even 4.5 years after a rupture. Don and colleagues[12] showed an association between consistent deficits in plantar flexor eccentric contraction and increased ankle dorsiflexion 2 years after an injury. In the present study, we observed decreased functional stiffness in end-range eccentric loading in the injured leg, with no association with the normalized Achilles tendon length. These results support the speculation that the eccentric capacity of the plantar flexors may influence the biomechanical changes occurring in the end of the second rocker.[12] Patients with more than a 30% difference between the injured and uninjured legs in the LSI of heel-rise height at 1-year follow-up have been shown to exhibit reduced heel-rise power at 6-year follow-up, which was associated with increased eccentric plantar flexor power during gait.[10] The patients in the current follow-up exhibited an LSI of heel-rise height between 69% and 72% at 1-year follow-up[5] and similarly increased eccentric plantar flexor power during gait at medium-term follow-up.

Stiffness

Stiffness is an important functional parameter, as energy return from the Achilles tendon stretching during dorsiflexion is a contributor to total push-off power.[31] Functional stiffness includes a position-dependent component that stores and releases energy (passive stiffness) along with possible simultaneous muscular work.[11] Caution should therefore be applied when comparing functional stiffness to passive stiffness.

At 1-year follow-up, the patients included in this study exhibited decreased passive stiffness in the early and terminal parts of dorsiflexion,[7] consistent with several previous studies.[9,19,28] Decreased energy storage has also been proposed to be caused by increased stiffness of the actual tendon.[1] The explanation to measure passive stiffness should then be the result of a longer, more slack tendon and therefore less stretching of the actual tendon structures during ankle dorsiflexion.[28] If the tendon is stretched to a lesser degree, energy storage in the Achilles tendon would be less,[12,19,22] and energy return to the push-off would be reduced. Decreased stiffness was also reflected in the smaller plantar flexor impulse and less work in the injured leg; however, peak positive power (push-off power) was not different between the legs.

At midterm follow-up, decreased quasi-stiffness was identified in the injured leg in the initial and late phases of dorsiflexion during the second rocker, which are the early and terminal parts of dorsiflexion. Reduced stiffness in the terminal phase, when tibial progression is mainly controlled by eccentric contraction in the triceps surae, could be related to the previously discussed decrease in eccentric capacity.

Reduced dynamic stiffness may also have implications for joint positioning, as the triceps surae and Achilles tendon play a large part in ankle proprioception.[9] Ankle proprioception is reduced in patients recovering from ATRs,[9] which could make them more susceptible to perturbations during gait. A recent study on healthy participants concluded that walking in unstable or randomly perturbating shoes increases co-contraction around the ankle joint and thereby decreases functional stiffness during loading.[3] This mechanism could help explain decreased quasi-stiffness in the initial phase of dorsiflexion. However, slippery or uneven surfaces have also been suggested to increase quasi-stiffness,[13] so further studies are warranted.

One-Leg Standing Balance

Our study hypothesis was that potential proprioceptive deficits after ATRs would increase sway length in the injured leg owing to the diminished sense of tendon length changes influencing ankle joint control.[30] Yet, no differences were found in sway length, rambling, or trembling. An explanation could be that sway length may not be associated with range of dorsiflexion but range of plantar flexion[18]; thus, increases in passive dorsiflexion and tendon length may not influence standing balance as hypothesized. Interestingly, a relation was found in the injured leg, in which an increase in quasi-stiffness was associated with an increase in sway length. This association was also present in the uninjured leg but was not significant. A larger degree of co-contraction has been speculated to result in increased sway length,[25] and as stated above, quasi-stiffness increases in unstable conditions. Yet, increased co-contraction is, as stated above, indicated to decrease quasi-stiffness. Further studies are needed to fully determine this connection.

Limitations

A limitation to the results found in the current study was the only partial inclusion of the original randomized groups. Participants in the original RCT who did not respond to the invitation for a medium-term follow-up may have been the patients with the best outcome from the initial treatment.

CONCLUSION

The current study showed that patients treated nonoperatively have a small increase in peak dorsiflexion along with decreased total internal plantar flexor moment and concentric plantar flexor power, as well as increased eccentric power with a delayed peak. Furthermore, we showed decreased quasi-stiffness in initial dorsiflexion and end-range dorsiflexion during gait. This study did, however, reveal that these deviations could not be directly associated with the measured tendon elongation.

The results on functional stiffness and sway indicate that there may be neuromuscular adaptations or compensations to structural and passive changes in the muscle-tendon complex after ATRs. Future studies may include muscle coordination as well as neuromuscular responses to perturbations or surface changes in the injured leg to understand sustained passive and active factors of gait and balance in midterm and long-term recovery after ATRs.

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